

**EGYPTIAN GUIDELINES ON
PREVENTION OF ATHEROSCLEROTIC
CARDIOVASCULAR DISEASE**

**THE EGYPTIAN HYPERTENSION SOCIETY WORKING GROUP ON
CARDIOVASCULAR PREVENTION GUIDELINES
DELTA-C PROJECT**

EXECUTIVE SUMMARY

PREPARED BY

M. MOHSEN IBRAHIM, MD

President of the Egyptian Hypertension Society
Director of Delta-C Project

2006

This Document has been approved by members from the Egyptian Hypertension Society, Egyptian Society of Cardiology, Egyptian Society of Nephrology, Egyptian Society of Diabetes and Egyptian Society of Obesity

TABLE OF CONTENTS

- Members of EHS Working Group and Advisory Board On Cardiovascular Prevention Guidelines
- Abstract
- Introduction
- Rationale for Egyptian guidelines
- What is special about Egyptian guidelines?
- Cardiovascular prevention strategies
- Assessment of cardiovascular risk
- Lifestyle modification
 - Smoking
 - Diet and nutrition
 - Physical activity
 - Obesity
- Major risk factors interventions
 - Hypertension
 - Dyslipidemia
 - Diabetes mellitus
- References and suggested readings
- Appendix

MEMBERS OF EHS WORKING GROUP AND ADVISORY BOARD ON CARDIOVASCULAR PREVENTION GUIDELINES

Chair: M. Mohsen Ibrahim, MD (Prof. of Cardiology, Cairo University)

Members of Writing Group (Alphabetical):

- | | |
|--|---|
| <ol style="list-style-type: none"> 1. Abdel Moniem Ibrahim, MD
Prof. of Physiology, Cairo University 2. Adel El-Etriby, MD
Prof. of Cardiology, Ain Shams University 3. Hossam Kandil, MD
Prof. of Cardiology, Cairo University 4. Hussien Rizk, MD
Prof. of Cardiology, Cairo University 5. Mahmoud Hassanien, MD
Prof. of Cardiology, Alexandria University 6. Mohamed Sobhy, MD
Prof. of Cardiology, Alexandria University 7. Omar Awwad, MD
Prof. of Cardiology, Ain Shams University | <ol style="list-style-type: none"> 8. Omar El-Khashab, MD
Prof. of Nephrology, Cairo University 9. Sameh Shahien, MD
Prof. of Cardiology, Ain Shams University 10. Samir Helmy Assaad, MD
Prof. of Endocrinology- Alexandria University 11. Sherif Hafez, MD
Prof. of Internal Medicine, Cairo University 12. Soliman Gharieb, MD
Prof. of Cardiology, Cairo University 13. Wafaa El-Aroussy, MD
Prof. of Cardiology, Cairo University 14. Wagdy Ayad, MD
Prof. of Cardiology, Alexandria University |
|--|---|

Assistant Staff:

- | | |
|---|--|
| <ol style="list-style-type: none"> 1. Dr. Hussien Heshmat
Cardiology Department, Cairo University 2. Dr. Amir Abdel Wahab
Cardiology Department, Cairo University | <ol style="list-style-type: none"> 3. Dr. Yasser Yazid
Cardiology Department, Cairo University 4. Dr. Karim Said
Cardiology Department, Cairo University |
|---|--|

Advisory Board (Alphabetical):

- | | |
|---|--|
| <ol style="list-style-type: none"> 1. Abdel Fatah Ferier, MD
Head of Cardiology Department,
Zagazieg University 2. Adel Zaki, MD
Prof. of Cardiology, Cairo University 3. Ahmed Abdel Latief Ramadan, MD
Prof. of Cardiology, Al-Azhar University 4. Ahmed Abdel Moniem, MD
Head of Cardiology Department,
Banha University 5. Ahmed Nassar, MD
Prof. of Cardiology, Ain Shams University 6. Ashraf Reda, MD
Prof. of Cardiology, Menofia University 7. Aziz Madkour, MD
Prof. of Cardiology, Al-Azhar University 8. Fathy Maklady, MD
Prof. of Cardiology, Suez Canal University 9. Fouad El-Nawawy, MD
Prof. of Medicine, Cairo University 10. Hany Abdel Razek, MD
National Heart Institute 11. Hassan Khalid, MD
Prof. of Critical Care Medicine,
Cairo University 12. Hazem Khamis, MD
Military Academy 13. Ibrahim El-Ebrashy, MD
Prof. of Medicine- Cairo University 14. Khaled Sorour, MD
Prof. of Cardiology, Cairo University | <ol style="list-style-type: none"> 15. Magdy Abdel Hamid, MD
Ass. Prof. of Cardiology, Cairo University 16. Medhat Ashmawi, MD
Prof. of Cardiology, Tanta University 17. Mokhtar Gomaa, MD
Prof. of Cardiology, Al-Azhar University 18. Nasser Taha, MD
Prof. of Cardiology, Menia University 19. Ramzy El-Mawardy, MD
Head of Cardiology Department,
Ain Shams University 20. Salah El-Ghazali, MD
Deputy Dean for Education and
Students Affairs,
Faculty of Medicine, Cairo University 21. Sameh Bakhom, MD
Ass. Prof. of Cardiology - Cairo University 22. Samir Abdel Kader, MD
Prof. of Cardiology, Assuit University 23. Sherif El-Tobgy, MD
Head of Cardiology Department,
Cairo University 24. Tarek Salah Khalil, MD
Head of Cardiology Department
Menofia University 25. Yasser Sharaf, MD
Prof. of Cardiology –Cairo University 26. Yehia Kishk, MD
Prof. of Cardiology, Assuit University |
|---|--|

ABSTRACT

- In Egypt and third world countries, there are no guidelines for prevention of atherosclerotic cardiovascular disease. Clinical guidelines developed in wealthy countries are inappropriate for most of the world population.
- Guidelines address both lifestyle modification and drug therapy for established risk factors. Because of limited resources, thresholds of pharmacologic interventions for treatment of hypertension and hypercholesterolemia and target levels were higher than in western guidelines.
- Two complimentary prevention strategies are needed. The first is directed to the whole population aiming at decreasing the risk profile of the whole community. The second is an individual approach targeting principally high risk individuals.
- Individuals at increased risk of developing future cardiovascular events should be the first target of the prevention programs. High risk individuals include patients with established atherosclerotic cardiovascular disease (e.g. angina, myocardial infarction, stroke), diabetic patients with additional risk factors, elderly individuals with risk factors, very high level of a single risk factor and multiple (> 3) risk factors.
- Complete smoking cessation is mandatory through both community and individual approaches.
- Both public and food industry should be aware of the hazards of unhealthy dietary style. A heart healthy diet should be made popular. This diet is low in animal fat and refined sugar and rich in fibers and unsaturated fat. Increased consumption of fruits, vegetables, whole grain, legumes and fish should be encouraged. Food labeling, government legislations and media campaigns are important tools.
- All individuals should be encouraged to establish and maintain at least 30 minutes of moderate intensity physical activity as 5 or more days/ week.
- A normal body weight (BMI 20-25 Kg/m²) and a waist circumference < 94 cm in men and < 80 cm in women are recommended. Control of obesity requires indefinite dietary and behavior therapy and regular physical activity. Combination of pharmacotherapy and lifestyle modification is more effective in weight loss than either approach alone.
- Blood pressure goal < 140/90 mmHg is recommended in all individuals; lower levels may be required in high risk individuals. Accurate blood pressure measurements should be encouraged in all routine office visits.
- Start of drug therapy for high BP, unless there is an emergency, should follow a period of monitoring and repeated measurements over a period varying from days to months depending upon BP level and global risk profile.
- In patients with dyslipidemia an elevated serum LDL-C should be confirmed on at least two separate measurements. Initiation of statin therapy is recommended only after failure of dietary intervention for a period of 3-6 months and should take into consideration the level of LDL-C and the patient's global risk profile. The use of statins for primary prevention is not recommended unless LDL-C is > 210 mg/dl, or if LDL-C >160 mg/dl in presence of multiple risk factors.
- Diagnosis of diabetes depends on accurate and repeated estimation of plasma glucose (PG). An optimal (normal) fasting PG is less than 100 mg/dl. If diet and exercise fail to normalize blood glucose within 3 months, oral therapy is initiated. Screening for hyperglycemia is indicated for individuals most likely to have impaired glucose tolerance, those with established atherosclerotic cardiovascular disease, hypertension and dyslipidemia.

INTRODUCTION

- The development of atherosclerotic (ASO) cardiovascular disease (CVD), namely coronary artery disease (CAD) and stroke is closely linked to the presence of a number of risk factors (RFs). These RFs when present will increase the chance of an individual for developing a CV event e.g. myocardial infarction, angina, stroke or coronary death.
- Many of the RFs are related to lifestyle and therefore can be modified while others are genetically determined and unmodifiable. Both genetic and environmental factors may co-exist.
- The chance of developing a future CV event are increased when more than one RF is present. A single RF unless of a very high level (e.g. very high BP or very high plasma cholesterol) is generally not enough to produce a CV event. However, there is a tendency of CV RFs to cluster i.e. more than one RF is usually present in the same individual.
- Eighty percent of coronary events are due to major modifiable RFs such as cigarette smoking, diabetes, hypertension, dyslipidemia (increase in plasma LDL-cholesterol and/or decrease in plasma HDL-cholesterol), obesity, sedentary lifestyle and psychosocial stress. Unmodifiable RFs include advancing age, male gender and a strongly positive family history of premature ASO CVD.
- Prevention of ASO CVD and delaying the development of CV events is possible through control and treatment of modifiable CV RFs.
- Although the outcome of prevention is not immediate like the results of treating acute conditions, yet on the long run it is more cost effective. Controlling hypertension and dyslipidemia, tobacco elimination and healthy diet improves disability, delays or prevents development of coronary events, stroke and death. Consequently the need of dialysis, and coronary revascularization will decrease.
- Prevention is better than cure, and when possible we should help people avoid developing heart disease in the first place.

RATIONALE FOR EGYPTIAN GUIDELINES

- A number of guidelines on prevention of ASO CVD are developed by western and international societies.
- In third world countries and in economically disadvantaged communities there are no cardiovascular prevention guidelines.
- National and regional guidelines are affected differently by considerations of cost, priorities in health care and prevalence rates of different cardiovascular risk factors.
- Clinical guidelines for the prevention of cardiovascular diseases developed in wealthy countries are inappropriate for most of the world's populations.
- In rich countries, where there is a viable health insurance system and various payment organizations, guidelines particularly for initiation of prevention drug therapy are largely "science based" and cost considerations are given less attention.
- In developing countries, resources for clinical prevention are severely limited. In these countries, cardiovascular disease prevention, if necessity, must give way to other health priorities.
- Guidelines for developing countries should, therefore, make a compromise between the optimal science based approach and the minimal affordable policy.
- Furthermore, there are difference in racial susceptibility, national life style and prevalence of cardiovascular risk factors among different countries.

NEED FOR EGYPTIAN GUIDELINES

1. In Egypt, resources for primary prevention are severely limited.
2. Cost considerations are given priority since largely science-based guidelines (USA and Europe) are unaffordable. Risk factors treatment thresholds, goals and use of drug therapy should, therefore, be modified. Egyptian national health care system will not pay for expensive drugs used in primary prevention, even in high-risk individuals. Therefore, recommendations for long term prevention based on guidelines from USA or Europe can not be accepted because of cost consideration.
3. Available cardiovascular risk predictive equations and scoring systems (e.g. Framingham, SCORE) are tedious and may not be applicable equally to Egyptian population.

WHAT IS SPECIAL ABOUT EGYPTIAN GUIDELINES

- Three factors were considered in the development of the Egyptian guidelines. These influenced the scope, dimensions, and many of the statements in the document:
 - A. Population characteristics.
 - B. Egyptian government and health system policies.
 - C. Egyptian environment.

A. Population Characteristics

1. Low income and high rates of poverty (average income per capita in Egypt is low).
2. High illiteracy rates particularly among rural women.
3. High prevalence rates of some cardiovascular risk factors (CV RFs) e.g. cigarette smoking, obesity, hypertension and diabetes (Egyptian NHP 1991-1994).
4. Misinformation about health and diet:
 - a. Obesity is a sign of good health, prosperity and sexual attraction among some Egyptian women.
 - b. Red meat is essential for energy, resistance to disease and sexual vigour.
5. Increased psychosocial stress due to overcrowding, unemployment and poor housing will create negative emotions of depression, anger and hostility. When these are complicated by illiteracy, there will be difficulty in health education, lifestyle modification and behaviour changes.
6. Cultural and social traits make involvement of women in exercising or physical activity sometimes unacceptable.

B. Egyptian Government and Health System Policies

1. Resources for primary prevention are limited.
2. Priority in spending is given to care of acute conditions.
3. Concept of RFs and CV prevention is not well understood among health professionals. Medical school training provides limited information in these areas. RFs scoring charts and tables are not known by the majority.
4. Health care system can not afford to pay for expensive drugs such as statins and ACE-Is used in either primary or secondary prevention.
5. Legislations against smoking and harmful palm oil use do not exist or are difficult to implement.
6. Majority of Egyptians purchase medications out of pocket.

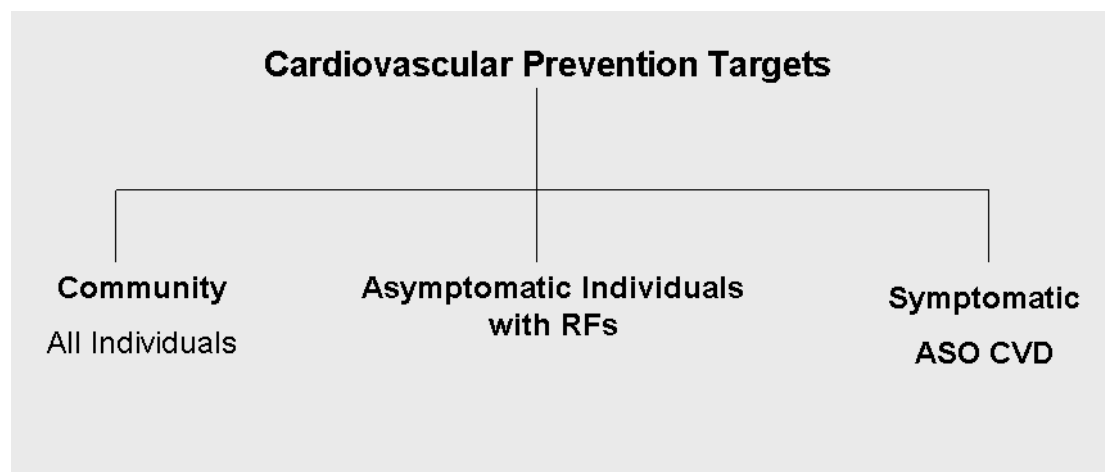
C. Egyptian Environment

1. Healthy foods such as fruits, vegetables, legumes (lentils and beans) and whole grain bread are available at reasonable prices (see appendix).
2. Mild weather conditions will encourage out-door sports and physical activity.
3. Religion has a very strong influence and can shape the behaviour of the public in right direction if it has the correct information.

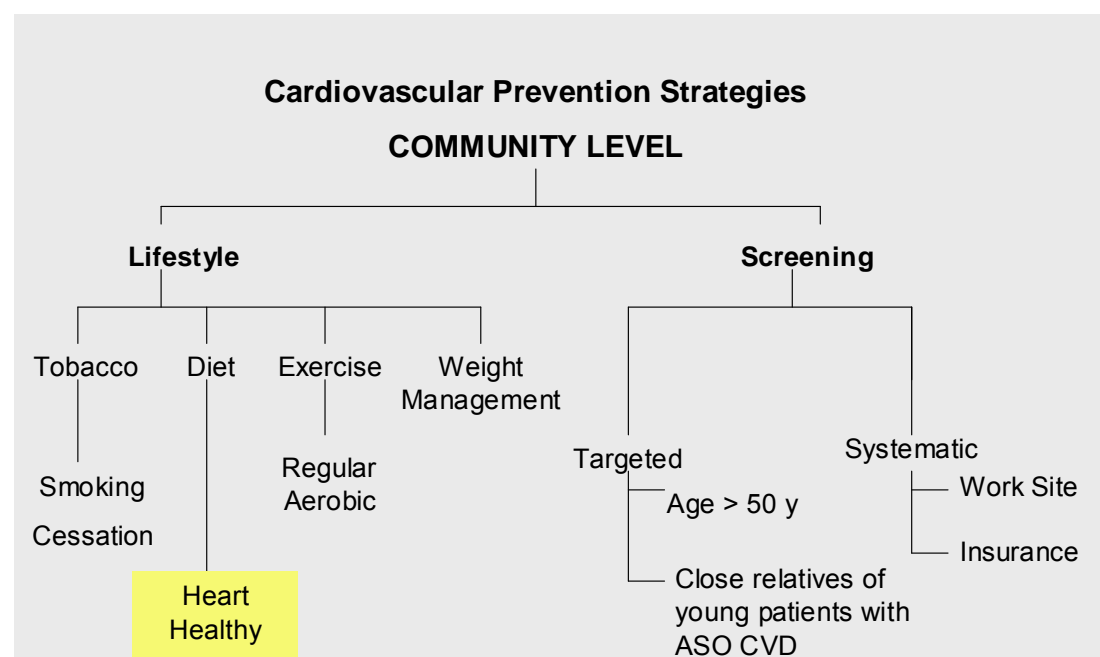
Scope of Guidelines and Main Features

1. Lifestyle change, public awareness and education received high priority.
2. Dietary approaches were stressed and given more space because of the affordable healthy foods in Egyptian environment (see appendix).
3. Community and public health prevention strategies were reinforced and outlined whenever possible while addressing lifestyle and major risk factors modifications.
4. Thresholds for initiation of pharmacologic therapy and targets of risk factors levels were higher than those reported in Americans or European guidelines. We realize that this may not be a scientific approach, yet there is no point in designing guidelines that are not applicable and not affordable to the majority of the population. There is strong evidence that some lowering in blood pressure (BP) is better than no change; similarly even small reduction in plasma LDL-C can be beneficial. At both the individual level and health care system, the long-term prescription of expensive drugs is doomed to fail. Discontinuation of drug therapy and lack of compliance are very common and a main reason is the cost.
5. Risk assessment was based on the number of risk factors and presence of established ASCVD. Charts, tables and risk scoring systems were not tested in the Egyptian population and the majority of physicians are not familiar or aware of them.
6. High prevalence rates of hypertension, obesity, cigarette smoking and diabetes among Egyptians dictated a more detailed discussion of these risk factors in the guidelines.

CARDIOVASCULAR PREVENTION STRATEGIES



- There are two types of CV prevention strategies, one is addressing the whole population where the aim is to lower the incidence of CV RFs in the whole community (public health or population strategy) and the second is directed to individuals at high risk (individual or high risk strategy).
- *The population strategy* depends on public education of healthy lifestyle, government legislations to develop a healthy smoke-free environment and promotion of healthy food through co-operation with food industry.
- *The high risk individual strategy* depends upon the evaluation of individuals seen in clinical practice, identification of persons at increased CV risk and treating the modifiable RFs. The aim of primary prevention is to identify early those individuals who are at risk of developing ASO CVD.



ASSESSMENT OF CARDIOVASCULAR RISK

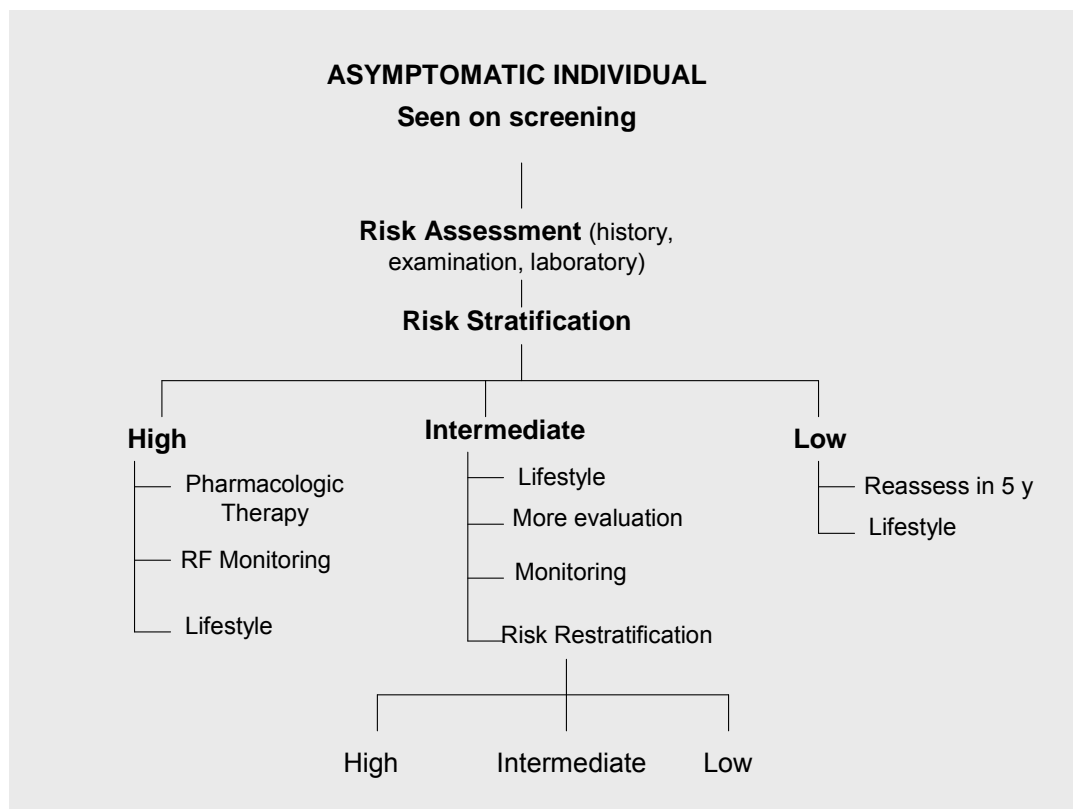
- Individuals at increased risk of developing future CV events should be the first target in any CV prevention program.
- Risk assessment depends on taking a detailed history of current or previous CV events (angina, infarction, claudication, transient ischemic attacks), family history of premature ASO CVD in first degree relatives, history of smoking, diabetes, hypertension (HTN) and physical activity. Presence of obesity (particularly the abdominal type characterized by increased waist circumference), presence of manifestations of ASO disease (e.g. peripheral artery disease, abdominal aortic aneurysm, carotid bruits) and signs of left ventricular hypertrophy (LVH) should be carefully searched for (table 1 and 2).

When laboratory facilities are available an electrocardiogram (ECG), urine examinations for proteinuria and blood tests for sugar, lipid profile (total cholesterol, LDL-C, HDL-C) and creatinine are performed (table 2).

Table (1): Atherosclerotic Cardiovascular Risk Factors

Risk Factor	Criteria
▪ Age	> 50 years in males and > 60 years in females
▪ Hypertension	No treatment SBP \geq 160 mmHg and/or DBP \geq 95 mmHg on 3 measurements. Treatment SBP \geq 140 mmHg and/or DBP \geq 90 mmHg
▪ Cigarette Smoking	Current or during the past 5 years
▪ Hyperlipidemia	TC \geq 240 mg, LDL-C \geq 160 mg/dl on 2 measurements
▪ Low HDL-C	< 40 mg/dl on two measurements
▪ Diabetes Mellitus	FPG \geq 126 mg/dl or PPG \geq 200 mg on 2 measurements
▪ Obesity	BMI \geq 30 kg/m ²
▪ Family History (first degree) of premature ASO CVD	Female relatives < 60 years, male relatives < 50 years
▪ Raised Serum Creatinine	> 1.6 mg/dl
▪ Left Ventricular Hypertrophy	ECG criteria

- The role of the rapidly growing list of evolving or non-traditional risk factors in primarily prevention is not established. These include lipid abnormalities (lipoprotein(a), small LDL-C and HDL-C particles), coagulation abnormalities (platelets function, plasma fibrinogen and other coagulation factors), metabolic defects (hyperhomocysteinemia), inflammatory markers (hsCRP and interleukin-6) and air pollution.



- Based upon the results of history, clinical and laboratory evaluation individuals can be categorized into 3 groups:
 1. *High risk* where the probability of developing a CV event in the coming 5 or 10 years is very high. The threshold for high-risk based on fatal cardiovascular events is defined as $\geq 5\%$ in 10 years (European Society of Cardiology Guidelines).
 2. *Intermediate risk* where the chances of developing an event is less than the high risk group.
 3. *Low risk* includes individuals who have an optimal cardiovascular risk profile i.e. young age, ideal body weight, normal BP ($\leq 130/80$ mmHg), optimal plasma lipid profile (LDL-C <130 mg/dl and HDL > 50 mg/dl) and no history of smoking or diabetes.
- *High risk* individuals should be identified and treated aggressively through lifestyle modification and pharmacologic therapy.
- The following individuals belong to the high risk category:
 1. Established ASO-CVD:
 - a. Coronary: MI, angina, revascularization procedures (CABG, PCI).
 - b. Cerebrovascular: stroke, TIA
 - c. Peripheral arterial disease.
 - d. Abdominal aortic aneurysm.

2. Diabetes mellitus in patients above the age of 50 years when there is microalbuminuria or additional RFs.
 3. Elderly individuals (> 65 y) with more than one major RF (e.g. hypertension, dyslipidemia, cigarette smoking, obesity, positive family history).
 4. Very high level of a single RF e.g. BP \geq 180/110 mmHg, LDL-C \geq 240 mg/dl, total cholesterol \geq 320 mg/dl.
 5. Multiple RFs (more than 3 of atherosclerotic cardiovascular risk factors in table (1)).
- *Intermediate risk:*
 1. Multiple RFs (2-3 of the previous RFs)
 2. Elderly individuals (> 65 y) with no or a single RF.
 3. Presence of the metabolic syndrome (abdominal obesity i.e. waist circumference > 94 cm in men and > 80 cm in women in addition to two or more of the following: fasting blood sugar > 110 gm/dl, BP > 130/80 mmHg, plasma triglycerides > 150 mg/dl or HDL-C < 40 mg/dl).

In the intermediate risk group, further evaluation is optional for risk restratification. This includes blood tests for inflammatory markers (hsCRP), urine for microalbuminuria, ECG-stress testing, echocardiography for LVH, imaging for coronary calcification and for carotid intimal-medial thickness and measuring the ankle-brachial pressure differences. Because of their cost, these tests are seldom performed.

- *Low risk:*
Individuals in this low risk category had no RFs or just one RF of mild to moderate intensity.
- *Factors contributing to increased CV risk:*
 1. Sedentary lifestyle and lack of physical activity.
 2. Increased psychosocial stresses: poverty, unemployment, social isolation, poor family relations and stressful job conditions.
 3. Impaired glucose tolerance and raised triglyceride levels.

Table (2): Assessment of Cardiovascular Risk

History	Physical Examination	Laboratory
▪ Angina	▪ Obesity	▪ ECG
▪ MI	▪ Waist Circumference	▪ Blood Sugar
▪ Stroke	▪ BP	▪ Lipid Profile
▪ TIA	▪ LVH	▪ S. Creatinine
▪ PCI, CABG	▪ Abdominal Aorta	▪ Microalbuminuria
▪ Claudication	▪ Peripheral Arteries	
▪ Family history	▪ Carotid Bruits	

- Smoking

Risk Scoring Systems

- Another approach for CV risk assessment based upon epidemiologic studies in USA (Framingham) and Europe (SCORE) using risk charts and assigning a number of points for each RF. This approach is not practical or familiar to Egyptian community, furthermore, it was not tested in the Egyptian population.

Methods for Prevention of ASO CVD

1. Lifestyle modification.
2. Major risk factors interventions.

LIFESTYLE MODIFICATION

SMOKING

- Tobacco use remains the single most preventable cause of death.
- Individuals and patients at any age can benefit from quitting smoking. Benefit on CV risk may take several years before it is seen.
- Smoking status should reflect life time exposure to tobacco and is not simply current tobacco use. Smokers who have only stopped recently (< 5 years) should be assessed as still smoking.

GOALS

- Complete smoking cessation, not simple reduction. Smoking as few as 5 cigarettes a day has an increased risk.
- Ensure a smoke-free environment with avoidance of passive smoking.

APPROACHES

Community Approach

1. Increase the price of cigarettes by increasing taxes.
2. Ban smoking in public places through clean indoor air legislation.
3. Disseminate through media an antismoking campaign.
4. Ban tobacco advertising and promotions.

5. Prevent selling tobacco to minors.
6. Provide smoking cessation aids through subsidizing nicotine replacement.
7. Youth focused programs.
8. Establishing smoking cessation clinics.

Individual Approach

A. Counseling, behavioral and psychosocial support

1. Always ask about history of smoking.
2. Brief and supportive advice on smoking cessation as often as possible.
3. Encouragement and motivations, learn techniques of distraction and relaxation.
4. Avoid situations that trigger smoking.
5. Provide appropriate facts on risks of smoking and educational material about how to quit.

B. Pharmacotherapy

1. Nicotine replacement therapy (NRT)

- Should be considered in individuals smoking > 10 cigarettes per day and fail to stop smoking in spite of repeated trials and in those with severe nicotine addiction.
- Nicotine patch provides consistent levels of nicotine delivery and is the agent of choice. It is recommended for 6 to 10 weeks. It is available in 7, 14 and 21 mg doses for single use per day, applied at night and left for 24 hours.
- Other forms of NRT which are short acting include gum, lozenges, and nasal spray.
- NRT patches are not recommended in patients with recent MI, unstable angina, severe arrhythmias or recent cerebrovascular events.

2. Psychotropic drugs

- Bupropion (Zyban): an antidepressant prescribed in 150 mg daily dose as sustained release capsules. It acts by decreasing both the craving for cigarettes and the symptoms of nicotine withdrawal.

It should be started at least 1 week before the cessation date and to continue for 2 to 3 months after the cessation date.

3. New drugs: Rimonabant

A cannabinoid receptor inhibitor that blocks the reinforcing effects of nicotine.

DIET AND NUTRITION

- Continued consumption of diet rich in saturated fats, cholesterol and salt is associated with increased risk for development of ASO CVD.

- Diet is the first treatment recommendation in patients with hypertension, diabetes, hyperlipidemia and obesity.
- A healthful dietary pattern is associated with a reduction in CVD events and risks.

GOALS

- Establish and maintain a healthy eating habit through:
 1. Substitute unsaturated fats for saturated and transfats.
 2. Increase consumption of omega-3 fatty acids.
 3. Consume diet rich in fruits, vegetables, nuts and wholegrain.
 4. Limit salt (Na Cl) intake when there is risk of high BP.

APPROACHES

Individual Approach

- Individuals should be aware of the composition of a heart healthy diet (table 3).
- The following dietary components are the targets of the dietary approach;
 1. Fats.
 2. Carbohydrates.
 3. Fibers.
 4. Salt.

1. Fatty food

- Limit fatty meat and dairy products (butter, creams, full cream cheese).
- Use skimmed or low fat milk, cottage cheese and low fat yogurt.
- Replace palm oil with olive/ soya/ corn or safflower oil.
- Avoid completely organ meat (e.g. liver, kidney, brain) and processed meat (such as sausages, salami and hamburger). Remove visible fat from meat and chicken skin before cooking.
- When cooking avoid frying, use boiling, steaming, baking or grilling.
- Do not use hydrogenated plant oils e.g. margarine.
- Nuts e.g. almonds, peanuts etc, are rich in monounsaturated fats and should be encouraged in moderation.
- Fat in fish is rich in omega-3 fatty acid and fish consumption is encouraged. Eat fish at least twice a week.

2. Carbohydrates

- Substitute complex carbohydrates and starches for simple and refined sugars.

3. Fibers

- Encourage a diet rich in soluble fibers.

- Fruits, vegetables, nuts, legumes (dried beans, peas and lentils), oats and wholegrain are rich in fibers.
- Increase fruit and vegetable intake to at least 5 to 9 servings/ day or 400-500 gm/day.
- Increase legume consumption to 30 gm/day. Soya bean is particularly recommended.
- Diet rich in oca, beans, oats and obergine can help in lowering plasma cholesterol.

4. *Salt* (sodium chloride)

- Restrict to less than 5 gm (1 teaspoon) per day.
- Reduce salt when cooking, limit processed and fast food.
- In patients with high BP, avoid salty cheese and preserved fish and meat.

Community Approach

- Food labeling is recommended in all food stores.
- Restaurants should be encouraged to serve a heart-healthy menus.
- Grocery stores and food markets should provide fruits, vegetables and grain products at a reasonable price.
- Food services at worksites should make available selections low in saturated fat and calories and provide a lot of fruits, vegetables and grain products.
- French bakery, and cookies should be discouraged.
- Production and distribution of plant based foods (vegetables, fruits and legumes) and grain based foods (preferably wholegrain) such as bread, pasta and rice should be encouraged.
- Media (TV, radio and press) should develop public awareness campaigns promoting heart healthy diet.
- At doctors' offices and other health facilities, printed materials (pamphlets, brochures) should be available outlining the risks of unhealthy diets and provide an outline of a heart healthy diet.
- Discourage eating meat.

Table (3): Heart Healthy Diet

1. Low animal fat:	Limit red meat and dairy products. Use low fat milk. Do not fry
2. More fibers:	Plenty of fruits, vegetables, legumes and whole grain
3. More unsaturated fat:	Olive oils, nuts, plant proteins and fish
4. Less refined sugars:	Limit candies and soft drinks
5. Less salt (Na Cl):	Avoid salty foods, canned, preserved food, fast food

PHYSICAL ACTIVITY

- Sedentary lifestyle is a major risk factor for ASO CVD. In women, it carries an increased risk for future CV events greater than obesity.
- Sedentary lifestyle is associated with a doubling of the risk of premature death.
- Increased levels of physical activity lead to improvements in BP, glucose intolerance, diabetes, HDL-C, triglycerides and obesity.
- Moderate physical exercise leads to a reduction in mortality.

GOALS

- All adults and children should engage in regular physical activity at a level appropriate to their capacity.
- Establish and maintain at least 30 minutes of moderate intensity physical activity on 5 or more days/week, these can be accumulated in shorter bursts of 10 minutes duration.

APPROACHES

Individual Approach

- Individuals who are not taking regular exercise should aim to take half an hour of moderate physical activity nearly every day.
- Encourage patients to be active depending upon the severity of disease.
- Simple exercises such as brisk walking, swimming and bicycling are ideal for all ages.
- Exercise intensity should increase to levels leading to an increase in heart rate, shortness of breath and sweating (not in hot environment).
- Begin at low intensity and gradually increase over several weeks, and gradually increase the time spent.
- All individuals should be encouraged to be physically active e.g. use stairs instead of elevator, walk to their worksite if possible or leave cars or buses at a long distance. Spend weekends walking in malls or parks and avoid sitting long hours in front of TV or computers.
- Daily physical activity should be part of the routine lifestyle.

Community Approach

- Schools and community organizations need to provide an environment that encourages and integrates physical activity into the daily lifestyle.
- Physicians and nurses need to emphasize the importance of physical activity as primary as adjunctive therapy for such medical conditions as hypertension, hypertriglyceridemia, glucose intolerance and obesity.

- Government and community organizations should promote the development of parks, sporting clubs, facilities for bicycling, swimming, etc.
- Media campaign educating public about the risks of sedentary lifestyle and benefits of simple physical activity.

OBESITY

- Obesity is a major independent RF for ASO CVD. There is a continuous, graded influence of BMI on CAD incidence even at below average weights.
- Obesity is a RF for diabetes, hypertension, dyslipidemia, sleep apnea and promotes a state of systemic inflammation.
- Obesity is assessed in terms of both BMI (BMI= body weight in Kg/ height in m²) and waist circumference (measured at a point midway between lower edge of rib cage and upper border of iliac rest).
- Obesity is defined as BMI ≥ 30 kg/ m² while overweight is BMI ≥ 25 Kg/ m².
- Waist circumference should be less than 80 cm in women and 94 cm in men.
- Central (visceral obesity- increased waist circumference) is a better predictor of future CV events and is more closely linked to impaired glucose tolerance, insulin resistance, higher BP and exaggerated dyslipidemia.
- Weight loss in obese persons results in improvement in BP, dyslipidemia and diabetes.

GOALS

- Maintain a normal weight (BMI 20-25 kg/m²) and a waist circumference < 94 in men and < 80 cm in women.
- Initial goal of weight loss therapy is to reduce body weight by approximately 10% from baselines.

APPROACHES

Individual Approach

- Encourage routine measurement of body weight and waist circumference.
- Assess and monitor both waist circumference and BMI.
- Set realistic targets of 5-10 kg weight loss for overweight and obese individuals. A reasonable time plan for a 10% reduction is 6 months.
- For weight loss to occur, it is necessary to use up more energy through regular physical activity and consume less calories from foods and drinks.
- Obesity, like a chronic disease, requires continuous monitoring and follow-up.
- Dietary therapy, physical activity and behaviour therapy should continue indefinitely.

- Main problem in obesity management is failure to maintain weight reduction after initial success.
- Behaviour therapy is needed. It includes self-monitoring, nutrition education, slow eating, physical activity and relapse prevention.
- Pharmacotherapy include:
 - a. Sibutramine which is an appetite suppressant.
 - b. Orlistat, an inhibitor of intestinal and pancreatic lipase that works by inhibiting fat absorption from the intestine.
 - c. Rimonabant, a cannabinoid receptor blocker which proved effective in large clinical trials but not yet available commercially.
- Drugs for obesity should be taken for long periods, for at least one year.
- Combination of pharmacotherapy and lifestyle modification is more effective in weight loss than either approach alone.
- Bariatric surgery has the best success in achieving weight loss but it carries an increased risk of operation morbidity and mortality. It is advised only in patients with BMI ≥ 30 kg/ m² associated with major obesity related co-morbidities or if BMI ≥ 40 kg/ m².

Community Approach

- Prevention of obesity should start in childhood. School and family environment should discourage the intake of rich caloric drinks and foods and stress the importance of regular physical activity.
- Breast feeding reduces the risk of developing obesity.
- Public awareness campaigns (media, social and sporting clubs) addressing the risks of overweight and providing lists of foods and drinks to avoid and the need for physical exercise.
- Establishing obesity clinics in central hospitals with a staff expert in diet and weight management.

- Reductions of 500 to 1000 calories a day are needed to produce weight loss at the recommended levels of 0.5 to 1.0 kg a week.
- Educate patients about the hazards of obesity.
- There are benefits of modest weight loss (5-10 % of body weight).
- Help patients set realistic goals.
- Emphasize gradual change in behaviour over time.
- Recommend caloric restriction and increased physical activity.
- Obesity is a chronic condition that requires lifelong treatment.

MAJOR RISK FACTORS INTERVENTIONS

HYPERTENSION

- An elevated BP ($\geq 140/90$ mmHg) on repeated measurements and on several office visits over a period of weeks to months constitute a diagnosis of hypertension.
- Risks of hypertension depend upon:
 1. Level of BP.
 2. Patient global cardiovascular risk profile.
- There is a linear relationship between BP level and future cardiovascular events. Risk starts at levels within what is considered to be the normal range and increases progressively with rise in BP.
- Risks of hypertension include coronary artery disease, heart failure, cerebrovascular disease, renal failure and dementia.
- High BP is commonly associated with other cardiovascular risk factors namely obesity, diabetes, impaired glucose tolerance and dyslipidemia.
- Management of hypertension depends upon:
 1. Establishing the presence of hypertension through accurate and repeated BP measurements.
 2. Assessment of patient's global cardiovascular risk profile through detailed clinical and laboratory evaluation.
 3. Need for pharmacologic therapy. The most central question in hypertension management is when to initiate drug therapy.
- Established dietary and lifestyle RFs for hypertension include: high salt (Na Cl) intake, low potassium (K) intake, overweight, obesity and excess alcohol drinking.

GOALS

- Achieve better BP control in established hypertensive patients. If targets BP levels can not be reached, some degree of BP reduction is better than no reduction.
- Detect hypertensive patients in the community particularly those with high cardiovascular risk profile e.g. patients with ASO CVD, diabetes, multiple risk factors.
- Ensure patients compliance with drug and non-pharmacologic therapy.
- For persons not at high risk, the BP goal is a level $< 140/90$ mmHg.
- Delay and/or prevent cardiovascular complications of hypertension.

APPROACHES

Individual Approach

- BP measurement in all routine office visits. If elevated and there is no emergency a period of observation and closer monitoring is needed before initiating drug therapy.
- Lifestyle modification is recommended in all hypertension patients, it includes:
 1. Lowered Na intake can reduce or obviate the need for antihypertensive medications. Restrict sodium intake to less than 2.4 g/day (< teaspoonful of salt).
 2. Weight reduction of around 5 kg in overweight patients can lower BP by 4.4/3.6 mmHg.
 3. Increased physical activity at least 30-45 minutes of aerobic activity on most days.
 4. Diet rich in potassium and fibers and low in dairy fat e.g. plenty of fruits and vegetables (> 5 servings/day), nuts (almonds, peanuts) and increase in vegetable protein intake from soya protein.

Pharmacologic (Drug) Therapy

The most central question in hypertension management is when to initiate drug therapy. Once treatment is started, it will continue indefinitely as there is no cure for established essential hypertension.

- Antihypertensive drug therapy should be initiated in the following conditions:
 - A. *Immediately* (regardless of risk stratification)
 1. Hypertensive emergencies.
 2. Very high BP levels(> 220/120 mmHg).
 - B. *After repeated measurements* over weeks to months. The duration of BP monitoring depends upon:
 - (1) BP level. (2) Risk profile. (3) Change in BP on follow up.
- Unless there are special indications, thiazide diuretics are the drugs of first choice in management of patients of mild to moderate hypertension (140-180/ 90-105 mmHg).
- Combination therapy (thiazide + beta blocker or ACE-inhibitor or calcium antagonists) is initiated in more severe degrees of hypertension (> 180/105 mmHg) or in high risk patients with mild to moderate hypertension.
- Once diagnosis of hypertension is established, life long monitoring of BP is required. Frequency of BP measurements depends upon BP level and risk profile. When BP is controlled (< 140/90 mmHg), regular check-ups every 3-4 months are recommended.

Table (4): Duration of initial monitoring depends on initial BP level and risk profile

Initial BP (mmHg)	Risk profile	Initial Follow up before initiation of therapy
≥ 180/110	High	Treat Immediately
	Low	Within 1 week
160-179 / 95-109	High	Within 2 weeks
	Low	Within 8 weeks
140-159 / 90-94	High	Within 8 weeks
	Low	Within 16-24 weeks

Need for Additional Drugs

- Aspirin: 75-150 mg/day aspirin is recommended in hypertensive patients above the age of 50 years, in presence of additional cardiovascular risk factor, provided that BP is adequately controlled (< 140/90 mmHg).
- Statins: small doses of statins e.g. atorvastatin 10 mg are added to antihypertensive therapy in patients with borderline elevation of LDL-C (> 130 mg/dl) and multiple RFs.

Community Approach

- Public awareness programs to encourage checking BP, stress the silent nature of high BP and the benefits of drug therapy.
- Public policy to encourage improvements in population eating habits. Stress the value of salt limitation and increasing fruit and vegetable consumption.
- Physician education regarding technique of accurate measurements of BP, initiation and monitoring of drug therapy.

DYSLIPIDEMIA

- An increase in plasma total cholesterol or LDL-C carries an increased risk for developing future coronary events.
- An elevated serum total cholesterol alone unless very high is a poor predictor of CAD risk, which is much increased when multiple RFs are present.
- Total cholesterol and HDL-C values should be the average of at least two measurements obtained from lipoprotein analysis on 12 h fasting sample.

- The increased CAD risk of elevated LDL-C starts at levels considered to be in the normal range (< 130 mg/dl) and the risk rises steeply with levels greater than 160 mg/dl.
- A reduced plasma HDL-C and/or increased triglycerides are also independent RFs for CAD.
- Total cholesterol / HDL-C or LDL-C / HDL-C ratios are very strong predictors of cardiovascular events.
- Lipid lowering therapy (e.g. statins) improved morbidity and mortality in patients with hypercholesterolemia, patients with CAD and diabetic patients.
- The decision to initiate drug therapy (statins) is determined by the patients global risk profile (i.e. presence of ASO CVD or other RFs), level of LDL-C and economic situation.
- Costs of cholesterol-lowering drugs appear to be a major limiting factor in management of patients for primary prevention.

FOOD COMPONENTS THAT RAISE LDL-C

1. Saturated fatty acids: present in full fat milk, fatty meats, palm oil and coconut oil.
2. Transfatty acids: present in partially hydrogenated vegetable oil, specially commercially prepared fried foods and cookies.

GOALS

- An LDL-C less than 160 mg/dl is recommended in all individuals. Levels less than 130 mg/dl and preferably less than 100 mg/dl are recommended in the high risk group.
- HDL-C levels more than 40 mg/dl and triglycerides less than 180 mg/dl are recommended.

It is not easy to identify a simple risk threshold at which lipid lowering therapy should be started.

APPROACHES

- Screening of dyslipidemia (12 hs fasting plasma lipid profile) is recommended in the following conditions:
 1. Clinically manifest ASO CVD.
 2. Diabetes.
 3. Hypertension.
 4. Very strong family history of premature CAD.

5. Skin xanthomas and xanthelasms.
- A cholesterol lowering diet (see section on Diet) and increased physical activity should be tried for a period of 3-6 months before initiating drug therapy (statins) in patients with hypercholesterolemia.
 - Patients at very high risk (e.g. acute coronary syndromes) including those with familial hypercholesterolemia may need drug therapy at an earlier stage.
 - Elevated serum LDL-C should be confirmed on at least two separate measurements.

INDICATIONS OF STATINS THERAPY IN PATIENTS WITHOUT CAD

1. In absence of any CV RFs:
 - Persistent LDL-C > 210 mg/dl in individuals > 40 years, in spite of dietary intervention
 2. In presence of one major CV RF
 - Persistent LDL-C > 190 mg/dl, despite dietary intervention.
 3. In presence of more than one major RF
 - Persistent LDL-C > 160 mg/dl, despite of dietary intervention.
 4. In presence of other established ASO CV disease or diabetes associated with other RFs
 - Persistent LDL-C > 130 mg, despite of dietary intervention.
- The first priority for lipid lowering therapy are patients with pre-existing ASO CVD.
 - Statins are the agents of choice for LDL-C lowering.
 - Statins therapy is recommended in the following conditions after trial of lifestyle treatment:
 1. All patients with CAD.
 2. High risk patients with LDL-C greater than 130 mg/dl.
 3. Intermediate risk patients with LDL-C greater than 160 mg/dl.
 4. Patients with familial hypercholesterolemia of LDL-C greater than 220 mg/dl.
 5. Individuals with a single RF and LDL-C greater than 190 mg/dl.
 - Initiate statins therapy at a small dose e.g. 10 mg atorvastatin, 20 mg simvastatin, 20 mg pravastatin, or 5 mg rosuvastatin unless there are special indications for aggressive statins therapy e.g. acute coronary syndromes. Increase the statin dose if , after 6 weeks, target LDL-C is not reached.
 - In resistant cases combine statins with drugs interfering with cholesterol absorption from the gut e.g. ezetimibe or resins. Both are expensive and can cause indigestion or interfere with drug absorption.

- Recommendations for primary prevention using cholesterol lowering drugs based on guidelines from the united states can not be accepted because of the realities of consideration of healthcare costs.
- Nicotinic acid is the drug of choice for treatment of low HDL-C.
- Fibrates are effective in lowering increased triglycerides levels.

Table (5): Recommended RFs Target Levels

	High Risk	Low Risk
BP (mmHg)	< 140/90	< 160/95
LDL-C (mg/dl)	< 130	< 160

Table (5): Drug Therapy Initiation Threshold

	High Risk	Low Risk**
BP* (mmHg)	≥ 160/95	≥ 170/105
LDL-C (mg/dl)	≥ 160	≥ 190

* On 2-3 consecutive office visits

** No other risk factors

DIABETES MELLITUS

- Diabetes is a major risk factor for CAD, strokes, peripheral arterial disease, renal failure, heart failure and blindness.
- Early and aggressive treatment of diabetes will prevent or delay the development of its complications.
- The diagnosis of diabetes depends upon accurate estimation of plasma glucose. Elevation of plasma levels should be confirmed by repeated testing on separate days. An increase in plasma glucose above the following cut points on repeated measurements will constitute a diagnosis of diabetes:
 1. Fasting plasma glucose (FPG) ≥ 126 mg/dl.
 2. Two hours plasma glucose after a glucose load of 75 gm glucose dissolved in water (2 hs PPG) ≥ 200 mg/dl.
 3. Symptoms of diabetes i.e. thirst, polyurea, loss of weight plus a random plasma glucose ≥ 200 mg/dl.
- Optimal (normal) FPG is less than 100 mg/dl and 2 hs PPG less than 140 mg/dl. Individuals with 2 hs PPG ≥ 140 mg/dl and < 200 mg/dl have impaired glucose tolerance.

GOALS

- Identify individuals with previously undiagnosed type 2 diabetes in the community.
- Maintain optimal blood sugar level in patients with diabetes ($HbA1c \leq 7\%$).
- Detection and control of other cardiovascular risk factors namely hypertension and dyslipidemia.

APPROACH

- Screening for hyperglycemia is indicated for individuals most likely to have IGT, who can benefit from prevention efforts and for the detection of undiagnosed diabetes. These are:
 1. Patients with established ASCVD: coronary, cerebral and peripheral arterial disease.
 2. Hypertension.
 3. Dyslipidemia.
 4. Overweight ($BMI \geq 25$ kg/m²) in individuals above the age of 45 years.
 5. Younger individuals with additional risk factors such as prior gestational diabetes, family history (first degree relatives) of type 2 diabetes, high risk ethnic and racial group.
- Measure plasma glucose on a fasting venous sample. If FPG is > 110 mg/dl, perform additional 2 hs postprandial test.
- First line management of hyperglycemia is in most cases, lifestyle intervention: physical activity, healthy eating and weight management. Then if required, addition of appropriate hypoglycemic therapy.

- If diet and exercise fail to normalize blood glucose within 3 months, oral therapy is initiated. Selection of the right agent should be individualized (Table 7).
- Tight glucose control with insulin for 3 or more months should be considered in patients with acute coronary syndromes.
- Insulin therapy is indicated, if there is severe hyperglycemia at diagnosis to overcome glucotoxicity. Insulin is given if hyperglycemia persists despite maximum dose of oral agent.
- Aggressive control of hypertension (i.e. BP < 130/85) and dyslipidemia (LDL-C < 130 mg/dl) is recommended in diabetic patients.
- In diabetic patients with multiple RFs, ramipril (10 mg/d) has a cardiovascular protective effect.

Table (7): Type 2 Diabetes: Current Treatment Options

Sulphonylureas	Stimulate insulin secretion by direct action on pancreatic β -cells
Meglitinides (prandial insulin releasers)	Stimulate insulin secretion, similar to sulphonylureas but with faster onset and shorter duration of action
Metformin	Improves insulin action by effects on insulin signaling and other mechanisms
Thiazolidinediones	Improve insulin action mainly by activation of PPAR- δ agonists
Acarbose	Slow rate of carbohydrate digestion by inhibiting intestinal α -glucosidase enzymes
Insulin	Decreases hepatic glucose output, increases glucose uptake and metabolism (especially skeletal muscle) and reduces lipolysis

Recommendations for Glycemic Control

- | | |
|--|---------------|
| HbA1C | < 7% |
| • Preprandial plasma glucose | 90 -130 mg/dl |
| • Peak post prandial plasma glucose | < 180 mg/dl |
| • Less intensive glycemic goals may be indicated in patients with severe or frequent hypoglycemia. | |

Source: American Diabetic Association

REFERENCES AND SUGGESTED READINGS

1. Agency for Health Care Policy and Research: The agency for health care policy and research smoking cessation clinical practice guidelines. *JAMA* 1996;275:1270-1280.
2. Alberti G: A desktop guide to Type 2 diabetes mellitus. European Diabetes Policy Group 1998-1999 International Diabetes Federation European Region. *Exp Clin Endocrinol Diabetes* 1999;107:390-420
3. American Heart Association, Comprehensive Risk Reduction for Patients with Coronary and Other Vascular Disease, *Circulation* 1995;92:2-4
4. Assmann G, Carmena R, Cullen P, Fruchart JC, Coronary heart disease: reducing the risk: a worldwide view, International Task Force for the Prevention of Coronary Heart Disease. *Circulation* 1999;100:1930-1938.
5. Assmann G, Cullen P, Jossa F, Coronary heart disease: reducing the risk the scientific backgrounds to primary and secondary prevention of coronary heart disease. A worldwide view. International Task Force for Prevention of Coronary Heart disease, *Arterioscler Thromb Vasc Biol* 1999;19:819-824
6. ATTP III Final Report II. Rationale for Intervention. *Circulation* 2002;106:3163.
7. Austin MA, Hokanson JE, Edwards K: Hypertriglyceridemia as a cardiovascular risk factor. *Am J Cardiol* 1998;81 (4A):7B-12B.
8. Avins AL, Neuhaus JM. Do triglycerides provide meaningful information about heart disease risk? *Arch intern Med* 2000; 160:1937-1944.
9. Baker GD, Ambrosioni E, Johnson KB. European guidelines on cardiovascular disease prevention in clinical practice. *European journal of cardiovascular prevention and rehabilitation* 2003; 10(suppl 1):S1- S78.
10. Behar S, Boyko V, Reicher-Reiss H, et al., Ten-year survival after acute myocardial infarction: comparison of patients with and without diabetes. SPRINT Study Group,. Secondary Prevention Reinfarction Israeli Nifedipine Trial. *Am Heart J* 1997;133:290-296.
11. British Cardiac Society, British Hyperlipidaemia Association, British Hypertension Society, Joint British recommendations on prevention of coronary heart disease in clinical practice, *Heart*, 1998;80 (supplement2): S1-S29
12. British Cardiac Society, British Hyperlipidemia Association, British Hypertension Society, British Diabetic Association: Joint British recommendations on prevention of coronary heart disease in clinical practice: Summary. *Br Med J* 2000;320:705-708
13. Brown L, Rosner B, Willett WW, Sacks FM: Cholesterol-Lowering effects of dietary fiber: a meta-analysis. *Am J Clin Nutr* 1999;69:30-42.
14. Burr ML, Gilbert JF, Holliday RM, Effects of changes in fat, fish, and fibre intakes on death and myocardial reinfarction: Diet and reinfarction trial (DART). *Lancet* 1989;II:757-761
15. Capewell S, Beaglehole R, Seddon M, et al Explanation for the decline in coronary heart disease mortality rates in Auckland, New Zealand, between 1982 and 1993, *Circulation* 2000; 102:15:1511.
16. Capewell S, Morrison CE, McMurray JJ, Contribution of modern cardiovascular treatment and risk factor changes to the decline in coronary heart disease mortality in Scotland between 1975 and 1994, *Heart* 1999; 81:3980.
17. Cater NB: Plant stanol ester: review of cholesterol-lowering efficacy and implications for coronary heart disease risk reduction *Prev. Cardiol* 2000;3:121-130
18. Clark MJ Jr, Sterrett JJ, Carson DS: Diabetes guidelines: a summary and comparison of the recommendations of the American Diabetes Association, Veterans Health Administration, and American Association of Clinical Endocrinologists. *Clin Ther* 2000;22:899-910
19. Clarke R, Frost C, Collins R: Dietary lipids and blood cholesterol: quantitative meta-analysis of metabolic ward studies. *Br Med J* 1997;314:112-117.
20. Cleland JG, Can improved quality of care reduce the costs of managing angina pectoris? *Eur Heart J* 1996;17:A29-A40.
21. Collins R, MacMahon S: Blood pressure, antihypertensive drug treatment and the risks of stroke and of coronary heart disease *Br. Med Bull* 1994;50:272-298
22. D'Agostino RB Sr, Grundy SM, Sullivan LM. et al, Validation of the Framingham coronary heart disease prediction scores: results of a multiple ethnic groups investigation *JAMA* 2001;286:180-187
23. David A., Cardiovascular Medicine at the National Heart and Lung Institute, Imperial College, London UK – Guidelines on Cardiovascular risk assessment and management, *European Heart Journal Supplements* (2005) 7 Supplement L) L5-L10 doi: 10.1093/eurheartj(sui079)
24. Despres J.P. and Others: Effects of Rimonabant on Metabolic Risk Factors in Overweight Patients with Dyslipidemia *NEMAG* 353 (20)2101-2208(2005) November 17, 2005
25. Despres JP. The insulin resistance-dyslipidemic syndrome of visceral obesity: effect of patients' risk. *Obes Res.* 1998;6 (Suppl 1):8S-17S
26. Durrington PN, Prais H, Bhatnagar D, et al. Indications for cholesterol-lowering medication: comparison of risk-assessment methods. *Lancet* 1999; 353:278-81

27. Ebrahim S, Davey Smith G Health promotion for coronary heart disease: past present and future *European Heart Journal* (1998) 19, 1751-1757.
28. Ebrahim S, Davey Smith G. Systematic review of randomized controlled trials of multiple risk factor interventions for preventing heart disease. *Br Med J.* 1997;314:1666-74.
29. Eckel RH, Krauss RM: American Heart Association call to action: Obesity as a major risk factor for coronary heart disease. AHA Nutrition Committee. *Circulation* 1998;97:2099-2100.
30. Family Heart Study Group, British Family Heart Study: its design and method, and prevalence of cardiovascular risk factors. *Br J Gen Pract* 1994;44:62-7
31. Field K, Thorogood M, Silagy C, et al. Strategies for reducing coronary risk factors in primary care: with is most cost effective? *BMJ* 1995; 310:1109-1112 (29 April)
32. Fletcher GF: How to implement physical activity in primary and secondary prevention . A statement for healthcare professionals from the Task Force on Risk Reduction, American Heart Association. *Circulation* 1998;96:355-357.
33. Francesco P. Cappuccio, Derek G Cook, Richard W Atkinson et al., Prevalence, detection, and management of cardiovascular risk factors in different ethnic groups in south London., *Heart* 1997;78:555-563
34. Frank B., Walter C, Willett, Optimal Diet for Prevention of Coronary Heart Disease *JAMA.* 2002;288:2569-2578
35. Galie N, and Other: Sildenafil Citrate Therapy for Pulmonary Arterial Hypertension *NEMAG* 353 (20)2101-2208(2005) November 17, 2005.
36. Grover SA, Paquet S, Levinton C, Coupal L, Zowall H. Estimating the benefits of modifying cardiovascular risk factors: a comparison of primary versus secondary prevention. *Arch Intern Med* 1998;158:655-662
37. Grundy M, Primary Prevention of Coronary Heart Disease Integration Risk Assessment with Intervention (*Circulation.* 1999;100:988-998)
38. Grundy M., Terry Bazzarre, James Cleeman et al. Prevention Conference V Beyond Secondary Prevention: Identifying the High-Risk Patient for Primary Prevention Medical Office Assessment . (*Circulation.* 2000;101:e3-e11)
39. Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and management of the metabolic syndrome: an American heart association/ national heart, lung and blood institute scientific statement. *Circulation.* 2005; 112:0000-0000
40. Grundy SM, Howard B, Smith S, et al., Prevention Conference VI: Diabetes and cardiovascular disease: executive summary: conference proceeding for healthcare professionals from a special writing group of the American Heart Association *Circulation* 2002;105:2231-2239.
41. Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto miocardico: Dietary supplementation with n-3 polyunsaturated fatty acids and vitamin E after myocardial infarction; results of the GISSI-Prevenzione trial. *Lancet* 1999;354;447-455.
42. Gueyffier F, Froment A, Gouton M: New meta-analysis of treatment trials of hypertension : improving the estimate of therapeutic benefit. *J Hum Hypertension* 1996;10:1-8.
43. Guy De Backer, Ettore Ambrosioni, Knut Borch-Johnsen et al. European guidelines on cardiovascular disease prevention in clinical practice *European Heart Journal* (2003)24, 1601-1610.
44. Gylling H, Miettinen TA: Cholesterol reduction by different plant stanol mixtures and with variable fat intake. *Metabolism* 1999;48:575-580.
45. Haffner SM, Lehto S, Ronnema T, et al., Mortality from coronary heart disease in subjects with type 2 diabetes and in non-diabetic subjects with and without prior myocardial infarction. *N Engl J Med* 1998;339:234.
46. Hansson L, Zanchetti A, Carruthers SG, et al. Effects of intensive blood-pressure lowering and low-dose aspirin in patients with hypertension: principal results of the Hypertension optimal Treatment (HOT) Study Group. *Lancet* 1998;351:1755-1762
47. Harris SB, Melzer SJ, Zinman B: New guidelines for the management of diabetes: a physician's guide. Steering Committee for the Revision of the Clinical Practice Guidelines for the Management of diabetes in Canada. *CMAJ* 1998;159:973-978.
48. Hobbs F.D.R. Guidelines and management of global risk: The European perspective *European Heart Journal Supplements Archive Vol. 6 Supp C (July 2004) Pp. 5-14 doi:10.1016/j.ehjsup. 2004.04.005*
49. Ibrahim MM, Appel LJ, Rizk HH. Cardiovascular risk factors in normotensive and hypertensive Egyptian. *Journal of Hypertension* 2001; 19:1-8
50. Ibrahim MM. RAS Inhibition in Hypertension. *Journal of Human Hypertension* (2006) 20, 101–108
51. Jack Froom, Paul Froom, Mignon Benjamin, Measurement and Management of Hyperlipidemia for the Primary Prevention of Coronary Heart Disease. *Journal of the American Board of Family Practice . Primary Prevention of Coronary Heart Disease.*
52. Jacques Genest, Jiri Frohlich, George Fodor Guidelines For The Management and Treatment Of Dyslipidemias, and Prevention of Cardiovascular Disease , jaques.genest@muhc.mcgill.ca

53. Jee SH, Suh I, Kim IS et al. Smoking and atherosclerotic cardiovascular disease in men with low levels of serum cholesterol. *JAMA* 1999; 282:2149
54. Jeffrey A., The effects of reducing sodium and increasing potassium intake for control of hypertension and improving health *Clin And Exper. Hypertension*, 21 (5&6) 769-783 (1999).
55. John Robsom and Gene Feder Predicting and reducing cardiovascular risk, *bmjournals.com* on 28 August 2005- Hear 2001;85:487-488
56. Juul-Moller S, Edvardsson N, Jahnmatz B, et al, Double-blind trial of aspirin in primary prevention of myocardial infarction in patients with stable chronic angina pectoris. The Swedish Angine Pectoris Aspirin Trial (SAPAT) Group. *Lancet* 1992;340:1421-1425.
57. Kamal WB. Blood pressure as a cardiovascular risk factor. *JAMA* 1996;275:1571-1576.
58. Kinosian B, Glick H, Garland G: Cholesterol and coronary heart disease: predicting risks by levels and ratios. *Ann Intern Med* 1994;121:641-647.
59. Klein S, Burke LE, Bray GA, et al. Clinical implications of obesity with specific focus on cardiovascular disease. *Circulation* 2004; 110: 2952-2967.
60. Krauss RM, Eckel RH, Howard B, et al. : AHA Dietary Guidelines: revision 2000: A statement for healthcare professionals from the Nutrition Committee of the American Heart Association. *Circulation* 2000;102:2284-2299.
61. Kris-Eterton PM, Yu S: Individual fatty acid effects on plasma lipids and lipoproteins: human studies *AM J Clin Nutr* 1997;65 (Suppl 5):1628S-1644S
62. Kuulasmaa K, Tunstall Pedoe H, Dobson A, et al. Estimation of contribution of changes in classic risk factors to trends in coronary-event rates across the WHO Monica Project populations. *Lancet* 2000; 355:675.
63. Law M: Plant Sterol and Stanol margarines and health. *Br. Med J*2000;320:861-864
64. Lemieux I, Pascot A, Couillard C, Lamarche B, et al, Hypertriglyceridemic waist. A marker of the atherogenic metabolic triad (hyperinsulinemia: hyperapolipoprotein B; small dense LDL) in men? *Circulation* 2000;102:179-184.
65. Lery D, Larson MG, Vasan rs, kannel WB, et al, The progression from hypertension to congestive heart failure *JAMA* 1996;275:1557-1562
66. Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study Group: Prevention of cardiovascular events and death with pravastatin in patients with coronary heart disease and a broad range of initial cholesterol levels. *N Engl J Med* 1998;339:1349-1357.
67. Luc F, Van Gaal, Ilse L, et al., What is the relationship between risk factor reduction and degree of weight loss?, *European Heart Journal Supplements* (2005) 7 (Supplement L.), L21-L26 doi: 10.1093/eurheart/sui082.
68. Marchioli R, Barzi F, Bomba E, et al., Early protection against sudden death by n-3 polyunsaturated fatty acids after myocardial infarction: time-course analysis of the results of the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto
69. Meltzer S, Leiter L, Daneman D, Gerstein HC et al: 1998 clinical practice guidelines for the management of diabetes in Canada. Canadian Diabetes Association. *CMAJ* 1998;159 Suppl 8:S1-S29.
70. Miettinen TA, Gylling H: Regulation of cholesterol metabolism by dietary plant sterols, *Curr Opin Lipidol* 1999;10:9-14.
71. Miettinen TA, Puska P, Gylling H, et al: Reduction of serum cholesterol with sitostanol-ester margarine in a mildly hypercholesterolemic population. *N Engl J Med* 1995;333:1308-1312.
72. Miocardico (Gissi)-Prevenzione. *Circulation* 2002; 105:1897-190.
73. National Institutes of Health: Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults-the evidence report, *Obes Res* 1998;6 Suppl 2:51S-209S.
74. National Institutes of Health: Executive summary of the clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. *Arch Intern Med* 1998;158:1855-1867.
75. Neal B, MacMahon S: The World Health Organization-International Society of Hypertension Blood Pressure Lowering Treatment Trialists" Collaboration: prospective collaborative overviews of major randomized trials of blood pressure- Lowering treatments. *Curr Hypertens Re.* 1999;1:346-356
76. Padwal R, Straus AE, McAlister FA. Cardiovascular Risk Factors and Their Effects on the Decision to Treat Hypertension: Evidence based review. *BMJ* 2001; 322:977-980
77. Padwal R. StrausSE; McAlister FA. Mulrow C, ed. Evidence-based hypertension London: BMJ Publishing Group,2001:33-38.
78. Pearson TA, Blair SN, Daniels SR et al.. Guidelines for Primary Prevention of Cardiovascular Disease and Stroke *Circulation* 2002
79. Pontiroli AE, Monti LD, Pizzini A, Piatti P: Familial clustering of arterial blood pressure, HDL cholesterol, and pro-insulin but not of insulin resistance and microalbuminuria in siblings of patients with type 2 diabetes. *Diabetes Care* 2000;23:1359-1364.

80. Randomised trial of cholesterol lowering in 4444 patients with coronary heart disease the Scandinavian Simvastatin Survival Study (4S). *Lancet*. 1994 344:1383-9.
81. Raw M, Anderson P, Batra A, et al. W: WHO Europe evidence based recommendations on the treatment of tobacco dependence. *Top Control* 2002;11:44-46.
82. Robert Beaglehole, Global Cardiovascular disease prevention: time to get serious : *Lancet* 2001;358:661-63
83. Rod Jackson, Carlene MM Lawes, Derrick A Bennett, Richard J Milne, et al. Treating Individuals 5 – Treatment with drugs to lower blood pressure and blood cholesterol based on an individual's absolute cardiovascular risk, *The Lancet* 2005;365:434-41
84. Rosolova H, Simon J, Community related life style intervention reduces coronary risk in the population, *European Heart Journal* (2000) 21, 1563
85. Scheuermann W, Razum O. Scheidt R et al. Effectiveness of a decentralized, community-related approach to reduce cardiovascular disease risk factor levels in Germany. *Eur Heart J*. 2000; 21:1591-7
86. Schillaci G, Vaudo G, Reboldi G, et al., High-density lipoprotein cholesterol and left ventricular hypertrophy in essential hypertension. *J Hypertens* 2001;19:2265-2270.
87. Scientific Statement Preventing Cancer, Cardiovascular Disease, and Diabetes: A Common Agenda for the American Cancer Society, The American Diabetes Association, and the American Heart Association
88. Scientific Statement Recommendations for Physical Activity and Recreational Sport. Participation for Young Patients With Genetic Cardiovascular Disease. Philip Greenland, Maria Deloria Knoll, Jeremiah Stamler et al, Major Risk Factors And Antecedents of Fatal and Nonfatal Coronary Heart Disease Events *JAMA*, August 20, 2003-vol 290, No 7
89. Sidney C, Smith Jr, Rod Jackson, et al, Principles for National and Regional Guidelines on Cardiovascular Disease Prevention A Scientific Statement from the World and Stroke Forum Doi:10.1161L01. CIR.oooo133427.35111.67
90. Sidney Smith C, Philip Greenland, Grundy M., Prevention Conference V Beyond Secondary Prevention: Identifying the High-Risk Patient for Primary Prevention. Executive Summary *Circulation*. 2000;101:111-116)
91. Smith GD, Song F, Sheldon TA, Cholesterol lowering and mortality: The importance of considering initial level of risk. *BMJ* 1993;306:1367-73
92. Stamler J, Greenland P, Van Horn L., et al: Dietary cholesterol, serum cholesterol, and risks of cardiovascular and noncardiovascular diseases. *Am J Clin Nutr* 1998;67:488-492
93. Stamler J, Stamler R, Neaton JD, Blood pressure, systolic and diastolic, and cardiovascular risks :US population data. *Arch Intern Med* 1993;153:598-615
94. Stampfer MJ, HU, Manson JE et. al. Primary prevention of coronary heart disease in women through diet and lifestyle. *N Engl J Med* 2000; 343:16.
95. Sterring Committee (Co-Chairs: Inoue S. and Zimmet P.) Redefining obesity and its treatment. Coordinated by the International Diabetes Institute, a world Health Organization Collaborating Centre for the Epidemiology, of Diabetes Mellitus and Health Promotion for Noncommunicable Diseases. Health Communications Australia Pty Limited. February 2002.
96. Stuart J Pocock, Calerie McCormack, Rancois Gueyffier. A score for predicting risk of death from cardiovascular disease in adults with raised blood pressure, based on individual patient data from randomized controlled trials, *BMJ* 2001; 323: 14
97. Surgeon General's report on physical activity and health. From the Centers for Disease Control and Prevention. *JAMA* 1996;276:522.
98. Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). Final Report. *Circulation* 2002;106:3143-3421.
99. Thomas A, Pearson, Erry L. et at, American Heart Association Guide for Improving Cardiovascular Health at the Community Level, A Statement for Public Health Practitioners, Healthcare Providers, and Health Policy Makers From the American A Heart Association Expert Panel on Population and Prevention Science. DOI:10.1161L01. CIR-0000054482-38437.13
100. U.S. Department of Agriculture and U.S Department of Health and Human Services: Nutrition and your health: dietary guidelines for Americans. Home and Garden Bulletin No. 232, Washington, D.C.: U.S. Department of Agriculture, 2000.
101. UK Prospective Diabetes Study (UKPDS) Group: Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes (UKPDS 38). *Br Med J* 1998: 317:703-713
102. US Preventive Services Task Force. Guide to clinical preventive services, 3rd ed, 2000-2002 [http:// www.ahrq. Gov/clinc/prevnew.htm](http://www.ahrq.Gov/clinc/prevnew.htm).
103. US Preventive Services Task Force. Guide to clinical preventive services, 3rd ed, 2000-2002 [http:// www.ahrq. Gov/clinc/prevnew.htm](http://www.ahrq. Gov/clinc/prevnew.htm).

104. US Public Health Service: A clinical practice guideline for treating tobacco use and dependence: A US Public Health Service report. The tobacco use and dependence clinical practice guideline panel, staff, and consortium representatives. *JAMA* 2000;284:3244-3254
105. Wadden T.A, and Others Randomized Trial of Lifestyle Modification and Pharmacotherapy for Obesity *NEMAG* 353 (20)2101-2208(2005) November 17, 2005
106. Wannamethee SG, Shaper AG, Ebrahim S: HDL-Cholesterol, total cholesterol, and the risk of stroke in middle-aged British men. *Stroke* 2000; 31: 1882-1888.
107. West R, McNeill A, Raw M: Smoking cessation guidelines for health professionals: an update. Health Education Authority. *Thorax* 2000; 55:987-999.
108. Williams RR, Hunt SC, Heiss G. et al., Usefulness of Cardiovascular Family History Data for Population-Based Preventive Medicine and Medical Research (the Health Family Tree Study and the NHLBI Family Heart Study). *Am J Cardiol* 2001;87:129-135
109. Wood D, De Backer G, Faergemant O, Prevention of coronary heart disease in clinical practice. Summary of recommendations of the Second Joint Task Force of European and other Societies on Coronary Prevention. *J Hypertension* 1998;16:1407-1414.
110. World Health Organization Rehabilitation and comprehensive secondary prevention after acute myocardial infarction Copenhagen. World Health Organization, 1983.
111. Yanovski S.Z. Pharmacotherapy for Obesity – Promise and Uncertainty *NEMAG* 353 (20)2101-2208(2005) November 17, 2005

APPENDIX (1)

قائمة طعام صحي لشخص وزن ٧٠ كيلو جرام و يمارس نشاط متوسط وتكلفتها بأحد الأحياء الشعبية

التكلفة (القرش)	السرعات الحرارية (kcal)	الوزن (جرام)	
			الإفطار
٥	٤٠٠	١٥٠	رغيف بلدى
٧٥	١٥٠	١٠٠	جبنة قريش بملعقة زيت ذرة صغيرة/
(٧٥)	(٢٥٠)	(٥٠)	أو طبق فول مدمس بزيت الذرة أو بصارة أو عدس أو بليلة
٢٥	٢٥	١٥٠	طبق سلطة صغيرة
٢٥	٦٥	١٥٠	برتقالة
٢٥	١٠٠	٢٥	كوب شاي بملعقة سكر صغيرة
١٥٥			التكلفة
			الغذاء
٥	٤٠٠	١٥٠	رغيف بلدى
(٢٥)	(٢٨٠)	(٢٥٠)	أو طبق بطاطس سمكة بلطى
٢٠٠	٢٠٠	(١٠٠)	أو علبة تونة صغيرة بملعقة زيت ذرة متوسطة
(٢٠٠)	(٢٠٠)	(٥٠)	أو طبق فول مدمس بزيت الذرة
(٧٥)	(٢٥٠)		أو طبق فول مدمس بزيت الذرة
٢٥	٢٥	١٥٠	طبق سلطة صغيرة
٢٥	٦٥	١٥٠	برتقالة
٢٥	١٠٠	٢٥	كوب شاي بملعقة سكر صغيرة
٢٨٠-١٧٥			التكلفة
			العشاء
٥	٤٠٠	١٥٠	رغيف بلدى
٧٥	١٥٠	١٠٠	جبنة قريش بملعقة زيت ذرة صغيرة
(٧٥)	(١٠٠)	(١٠٠)	أو علبة زبادى (خالى) الدهن بملعقة عسل اسمر
٢٥	٢٥		طبق سلطة صغيرة
٢٥	٦٥	١٥٠	برتقالة
٢٥	١٠٠	١٥٠	كوب شاي بملعقة سكر صغيرة
١٥٠		٢٥	التكلفة
(٥٢٥) ٥٨٠-٤٧٥	حوالى ٢٥٠٠		المجموع

APPENDIX (2)

A Systematic Approach to Management Based on BMI and Other Risk Factors
Assess Overall Health Risk from BMI and Other Risk Factors, e.g. Waist Circumference

BMI	Additional Risk Factors?	
25-29.9	Absent	—————→ Weight maintenance, healthy diet, exercise.
	Present	—————→ Goal for diet, exercise, behavior: primarily geared to risk management. Weight loss needed if risk not reduced substantially within 3 months, then, aim for 5-10 kg over 24 weeks by mild energy deficit. If not achieving this weight reduction at 24 weeks and risks persist, test usefulness of drug to reduce risk by weight management
30-34.9	Absent	—————→ Goal of 5-10 percent weight loss
	Present	—————→ Consider very low calorie diet and drug therapy if diet, exercise and lifestyle program unsuccessful after 12 weeks in reducing all risk factors
35-39.9		—————→ Use full therapy including drugs to achieve > 10% weight loss
≥ 40		—————→ Refer to specialists for separate management and consideration of surgery if conventional treatment fails. Aim for 20-30% weight reduction

Source: WHO. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. WHO Tech Rep Ser 2000; 894:i